

Mathematical Model for Infection and Removal

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ABSTRACT

The mathematical model of infectious diseases is a tool that has been used to study the mechanisms by which disease spread, to predict the future course of an outbreak and to evaluate strategies to control an epidemic. We envisaged a community of n individuals comprising at time t , x , susceptible, y infectious in circulation and z individuals who were isolated, dead, or recovered and immune. We further postulated infection and removal rates β and γ , so that there wave $\beta xydt$ new infections and γydt removals in time dt . The simplest way to do this is to introduce a birth parameter μ , so at to give μdt new susceptible in time dt . If the population is to remain stable the arrival of new susceptible must be balanced by an appropriately defined birth rate. The present paper represents the model in special way, in which the infection occurs in human's body then the resistance of body gradually decays same as motion decays in damped oscillation. On solving the equation of model, we get solution that gives the idea about the seasonal variation in infection.

KEYWORDS: Infection and Removal, Mathematical model, Seasonal variation

INTRODUCTION:

The mathematical model of infectious diseases is a tool that has been used to study the mechanisms by which disease spread, to predict the future course of an outbreak and to evaluate strategies to control an epidemic (Eisinger and Thulke, 2008; Parshani, et. al., 2010; Valdez, et. al., 2020;). We envisaged a community of n individuals comprising at time t , x , susceptible, y infectious in circulation and z individuals who were isolated, dead, or recovered and immune (Bailey, 1964; 1975). We further postulated infection and removal rates β and γ , so that there wave $\beta xydt$ new infections and γydt removals in time dt . An additional assumption to be made here is that the stock of susceptible is continually replenished (Barlett, 1956 and Brown, 1971).

The simplest way to do this is to introduce a birth parameter μ , so at to give μdt new susceptible in time dt . If the population is to remain stable the arrival of new susceptible must be balanced by an appropriately defined birth rate (Carrier, 1996; Cosma 2018; Sridhar and Majumder, 2020). The simplest model that can be constructed avoids explicit reference to a death rate by concentrating on the groups of susceptible and infective, the former at any rate being supposed not subject to death (Bodmer and Cavalli, 1976). This is equivalent to assuming that on average the deaths of removed individuals are just balanced by births of new susceptible (Maki, and Thompson, 1973).

How to cite this paper: Shukla Uma Shankar "Mathematical Model for Infection and Removal"

Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN: 2456-6470, Volume-5 | Issue-2, February 2021, pp.612-614, URL: www.ijtsrd.com/papers/ijtsrd38523.pdf



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MODEL DEVELOPMENT AND APPLICATION:

$$\begin{aligned} \frac{dx}{dt} &= -\beta xy + \mu \\ \frac{dy}{dt} &= \beta xy - \gamma y \\ \frac{dz}{dt} &= \gamma y \end{aligned} \quad (1.1)$$

Equilibrium values X_0 and Y_0 are given by equating the differential coefficients to zero.

$$\begin{aligned} x_0 &= \frac{\gamma}{\beta} \\ \text{Thus } y_0 &= \frac{\mu}{\gamma} \end{aligned} \quad (1.2)$$

The equations for small departures from these equilibrium values are obtained by writing

$$\begin{aligned} x &= x_0 (1+u) \\ y &= y_0 (1+v) \end{aligned} \quad (1.3)$$

and substituting (1.3) in (1.1)

$$\begin{aligned} \sigma \frac{du}{dt} &= -(u + v + uv) \\ \tau \frac{dv}{dt} &= u (1 + v) \end{aligned} \quad (1.4)$$

Where

$$\sigma = \frac{\gamma}{\beta} \mu, \quad \tau = \frac{1}{\gamma}$$

It we now neglect the product uv and eliminate u from the two equations (1.4) we obtain the second order differential equations in v .

$$\frac{d^2 v}{dt^2} + \frac{1}{\sigma} \frac{dv}{dt} + \frac{1}{\sigma \tau} v = 0 \quad (1.5)$$

$$D^2v + \frac{1}{\sigma} Dv + \frac{1}{\sigma\tau} v = 0$$

$$\text{or, } (D^2 + \frac{1}{\sigma} D + \frac{1}{\sigma\tau})v = 0$$

$$\text{So, } D = \frac{1}{2} \left(\frac{1}{\sigma} \pm \sqrt{\frac{1}{\sigma^2} - \frac{4}{\sigma\tau}} \right)$$

$$D = -\frac{1}{2\sigma} \pm \frac{i}{2} \sqrt{\frac{4}{\sigma\tau} - \frac{1}{\sigma^2}}$$

Solution of the equation (1.5) is

$$v = e^{\frac{-1}{2\sigma}t} \{C_1 \cos(\sqrt{\frac{4}{\sigma\tau} - \frac{1}{\sigma^2}} t) + C_2 \sin(\sqrt{\frac{4}{\sigma\tau} - \frac{1}{\sigma^2}} t)\}$$

with help of boundary condition for $\tau = 4\sigma$

$$\text{we have } v = v_0 e^{\frac{-1}{2\sigma}t} \cos\left(\sqrt{\frac{4}{\sigma\tau} - \frac{1}{\sigma^2}} t\right)$$

$$v_1 = v_0 e^{\frac{-1}{2\sigma}t} \cos \xi t \quad (1.6)$$

$$\text{Where, } \xi = \left(\sqrt{\frac{4}{\sigma\tau} - \frac{1}{\sigma^2}} \right)$$

for a suitably chosen origin of time, we then obtain the solution for u given by

$$u_1 = v_0 \left(\frac{\tau}{\sigma} \right)^{\frac{1}{2}} e^{\frac{-1}{2\sigma}t} \cos(\xi t + \psi)$$

$$\text{Where, } \cos \psi = -\frac{1}{2} \left(\frac{\tau}{\sigma} \right)^{\frac{1}{2}} \quad 0 \leq \psi \leq \pi \quad (1.7)$$

The solutions u_1 and v_1 , which are linearized or first order components only, clearly involve damped harmonic trains of waves with period $2\pi/\xi$. In this application to measles, τ equal to two weeks the approximate incubation period and, from the data available to him he estimated σ in London to be roughly 68.2 weeks. Equation (1.6) give the period $2\pi/\xi = 75.7$ weeks, with a peak to peak damping factor of $e^{\frac{-\pi}{\sigma\xi^2}} = 0.58$.

For somewhat larger oscillations we ought to take in to account the non-linear character of the equations (1.4). For this purpose it is convenient to write:

$$u = u_1 + a_{11}u_1^2 + a_{12}u, u_1 + a_{22}u_1^2 + \dots \quad (1.8)$$

Where the coefficient a_{ij} and b_{ij} , can be found in terms of $\frac{\tau}{\sigma}$ by straight forward substitution in (1.4). It $\frac{\tau}{\sigma}$ is small, so that $\psi - \pi/2$ we have approximately:

$$\left. \begin{aligned} u &= u_1 \left(1 + \frac{1}{3} v_1 \right) \\ v &= v_1 + \frac{1}{3} v_0^2 e^{\frac{-t}{2\sigma}} \cos 2\xi t \\ u_1 &= -v_0 \left(\frac{\tau}{\sigma} \right)^{1/2} e^{\frac{-t}{2\sigma}} \sin \xi t \end{aligned} \right\} \quad (1.9)$$

It will be noticed that the damping coefficient has relatively little influence on the period $\frac{2\pi}{\xi}$ which for small $\frac{\tau}{\sigma}$ is roughly $2\pi(\sigma\tau)^{\frac{1}{2}} = 2\pi\sqrt{\beta\mu}$.

And so largely depends on the birth rate for new susceptible and the infection rate.

As an alternative to the foregoing approximate discussion we can always investigate special cases by step by step

numerical solution of (1.1) through special care in needed to ensure sufficient accuracy and to avoid mistaken conclusion about the effect of damping.

Another way of representing the oscillatory behavior of the process is to plot the path traced by the point (x,y). This also allows a convenient method of comparing the deterministic solution with path followed by actual realizations of the stochastic analogue. It can be shown that the deterministic curve found in this way has the form of a spiral converging on the equilibrium point (x_0, y_0) , and that this occurs in spite of the non-linear form of (1.1).

An elegant argument due to G.E.H. Reuter (Bortlett, 1956) is as follows.

Consider the function

$$f(u, v) = \{(1+u) \cdot \log(1+u) + \left(\frac{\tau}{\sigma}\right) \{(1+v) \cdot \log(1+v)\} \quad (1.10)$$

Differentiating with respect to t and using (4)

$$\left(\frac{df}{dt} \right) = -\frac{u^2}{\sigma(1+u)} \leq 0 \quad (1.11)$$

Thus f continually decreases along any path for which t increases. Since $i \geq \frac{\tau}{\sigma}$, it follows that f tends to a finite limite for $f_0 \geq 1 + \frac{\tau}{\sigma}$ as $t \rightarrow \infty$. Now the curve $f = c$ are closed surround the point (x_0, y_0) and shrink down as $c \rightarrow 1 + \frac{\tau}{\sigma}$

By considering the second different coefficient d^2f/dt^2 we can show that $f_0 = 1 + \frac{\tau}{\sigma}$, so that the point (x, y) must actually tends to (x_0, y_0) .

An important consequence of the above discussion is that, while the additional of a constant influx of fresh susceptible is sufficient to account for epidemic waves a period of about the order of magnitude the damping down to a steady endemic state entailed by the calculations is at variance with observed epidemiological facts.

ESTIMATION OF SEASONAL VARIATIONS IN INFECTION RATE:

The simplest modification is to try replacing the infection rate β by $\beta' = \beta + \beta_1 \cos wt$.

Where, $2\pi/w = 52$ weeks.

We suppose the relative amplitude of these forced oscillations. $\rho = \frac{\beta_1}{\beta}$ to be small.

Substituting the new value of β in (1.1) and using (1.3) gives equations corresponding to (1.4). These can now be deal with as before by elimination u to yield to modified form of (1.5).

$$\frac{d^2v}{dt^2} + \frac{1}{\sigma} \frac{dv}{dt} + \frac{1}{\sigma\tau} v = -\frac{\tau\omega}{\tau} \sin\omega t \quad (1.12)$$

The particular integral of (1.12) representing the force oscillation term is by standard result of type.

$$V = A \cos(Wt + \epsilon)$$

$$\text{Where, } A = \frac{\sigma\omega}{\tau} \left\{ \left(\frac{1}{\sigma\tau} - \omega^2 \right) + \left(\frac{\omega}{\sigma} \right)^2 \right\}^{1/2} \quad (1.13)$$

Since the rate at which new notifications actually occur is given by γy , we have

$$\begin{aligned} \gamma y &= \mu(1+v) \\ &= \mu(1+v_1+v) \end{aligned} \quad (1.14)$$

Where v_1 is the first order solution (1.6). If we take $\tau = 2\sigma = 68.2$ as before the $\omega = \pi/2\sigma$, the putting in (1.13) gives A as approximately 8.1ρ .

This shows that 10% variation in β' , as envisaged by Soper, would lead to seasonal variations of about 80% in the rate of notifications.

ALLOWANCE FOR INCUBATION PERIOD:

Another possible source of error in the use of the simple continuous infection model is that it makes no allowance for the effect of a fairly well-defined incubation period, such as is clearly recognizable in measles (Ludwig, 1974 and Larger, 1976).

If we suppose that there is a latent period of length a after infection, at the end of which the infected individual becomes infectious, the processes of infection and removal then proceeding as before, the deterministic equations will be

$$\begin{aligned} \frac{dx(t)}{dt} &= -\beta x(t)y(t-a) + \mu \\ \frac{dx(t)}{dt} &= -\beta x(t)y(t-a) + \gamma y(t) \end{aligned} \quad (1.15)$$

The equilibrium values are exactly the same as for (1.1). If we now put $D = \frac{d}{dt}$, the equations for u and v are found to be.

$$\begin{aligned} \left(D + \frac{1}{\sigma}\right)u + \frac{1}{\sigma}e^{-aD}v &= 0 \\ -\frac{1}{\sigma}u + \left(D + \frac{1}{\sigma} - \frac{1}{\sigma}e^{-aD}\right)v &= 0 \end{aligned} \quad (1.16)$$

By short heuristic treatment of (1.16), using as operational method is as follows. If we assume that the infectious period is short, both β and γ are large and τ tends to zero.

The differential equations for v reduces to

$$\left\{\left(D + \frac{1}{\sigma}\right)e^{aD} - D\right\}v = 0 \quad (1.17)$$

The form of the solution depends on the roots of

$$\left\{\left(D + \frac{1}{\sigma}\right)e^{aD} - D\right\} = 0$$

If we write this as

$$aD = -\log\left\{\left(D + \frac{1}{\sigma}\right)\right\}$$

and expand the logarithm in negative powers of D .

We obtain

$$aD = -\frac{1}{\sigma D} + \frac{1}{2\sigma^2 D^2} - \dots \quad (1.18)$$

The first approximation to (1.18) is given by $D = \sqrt{-a\sigma}$ and the second

$$aD = -\frac{1}{\sigma D} + \frac{1}{2\sigma}$$

Which is equivalent to the equation

$$\frac{d^2\omega}{dt^2} + \frac{1}{2\sigma}\frac{d\omega}{dt} + \frac{1}{\sigma a}\omega = 0 \quad (1.19)$$

Comparing this shows that the approximate solution here is similar to the previous one, except that we now have a instead of τ , and the damping coefficient is halved.

RESULT AND DISCUSSION:

From the above discussion we see the model has been represented in special way, in which the infection occurs

in human body then the resistance of body gradually decays same as motion decays in damped oscillation (Kermack, and Kendrick, 1927; Hoppensteadt, 1975). On solving the equation of model we get differential equation of damped harmonic oscillation, which shows the phenomenon of occurrence of the infection. Their solution gives the idea about the seasonal variation in infection.

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